surgical complications would be, irrespective of the syphilis. A constricting tumor of the pylorus, for example, if followed by dilatation of the stomach and intractable vomiting, which does not yield to syphilitic medication alone, should be excised or a gastroenterostomy should be performed, even though syphilitic subjects are, as a rule, poor surgical subjects. Yet in these cases we should not hesitate any more than we would in cases of diabetes, when the indications are to save life, even in spite of the diabetes. When once the stomach symptoms have abated, the patient should be given to understand that repeated antisyphilitic cures are necessary, in order to avoid constitutional symptoms in the future.

THE LIVER IN TUBERCULOSIS.

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The liver in tuberculosis has been studied by a number of observers in the course of the last century. By the earlier observers it was thought to be only rarely the seat of tubercles or other manifestation of the general disease, but with the improvement of the microscope and histological methods, we find that very few cases of tuberculosis go to autopsy without some evidence of the disease in the liver.

Louis,[1] in 1843, found tuberculosis of the liver but twice in 120 autopsies on tuberculous subjects. Forster,[2] in 1854, was able, after a prolonged search, to find but three cases of hepatic tuberculosis. In an investigation undertaken by Bristowe,[3] in 1858, to discover the relation between liver abscess and intestinal ulceration, he found that of 167 cases of tuberculous ulcers of the intestine, but 17 showed "tuberculous cavities of the liver." Waldenberg,[4] in 1869, reported that it is the organ most frequently affected in experimental tuberculosis, but this is due to inoculation into the peritoneal cavity. Arnold,[5] in 1880, considered it an almost constant finding in cases of tuberculosis. Simmonds,[6] in 1888, found it in 76 per cent. in adults, in 92 per cent. in children, and in 78 per cent. in general. Zehden,[7] in 1897, found miliary tubercles in 50 per cent. of fatal cases, corresponding with the frequency of intestinal ulceration. Rolleston[8] is inclined to put it at a lower figure than 50 per cent. Rosenberger,[9]

[1] Recherches sur la phthisie, Paris, 1843.

[2] Handb. der spec. path. Anat., 1854.

[3] Trans. Brit. Path. Soc., 1858, ix, 241.

[4] Die Tuberculose, Berlin, 1869.

[5] Virchow's Archiv, 1880, Band. lxxxii.

[6] Deut. Archiv für klin. Med., 1888.

[7] Cntrlbl. für path. Anat., 1897, viii.

[8] Diseases of the Liver.

[9] Trans. Phila. Path. Soc., 1906, ix.

in 1906, found miliary tubercles in 83.8 per cent. of livers examined histologically, and White,[10] in 1907, found them in 70.3 per cent.

Tuberculosis manifests itself in the liver as: (1) Miliary tubercles; (2) solitary tubercles; and (3) tuberculous cirrhosis.

I. MILIARY TUBERCLES. Miliary tubercles are decidedly the most common manifestation of tuberculosis in the liver, and have been described by many observers. They are usually found either in or adjoining the portal spaces, but may be found within the lobule. It is thought by most men that the infection comes from ulcers of the intestine by way of the portal vein, and that the lesion is located in the portal spaces for that reason.

Infection of the liver may take place in utero, the bacilli being transmitted by the umbilical vein. Sabourad,[11] in 1891, found miliary tubercles of the liver and spleen in a child eleven days old, in whose mother there was phthisis pulmonalis, but no genital or mammary tuberculosis. He concluded that the infection travelled by way of the umbilical vein. Nocard,[12] in 1895, reported a case of tuberculosis of the liver in a calf, in whose mother's placental cotyledons there were tubercles containing giant cells. Von Honl[13] found tubercles in the liver and other organs of a fifteen-day-old child in whom the tubercles undoubtedly antedated birth. His reason for believing that they were older than fifteen days was the amount of fibrous tissue about the lesions. D'Arrigo[14] inoculated female guinea-pigs with tuberculosis, and then allowed them to become pregnant. Some went to term and others aborted. In those going to term the offspring showed tubercles, particularly of the abdominal organs. In those aborting after the first half of pregnancy there were tubercles and tubercle bacilli in the livers of the offspring. Bar and Renon[15] inoculated into animals the blood from the cut umbilical cord of children born of tuberculous mothers. In two cases out of five the animals showed tuberculosis. In one case the child died, and portions of its organs were inoculated into animals and produced tuberculosis in the animals. From the observations of these authors we conclude that tuberculosis can be carried from mother to child by way of the umbilical vein, and that it usually manifests itself in the liver.

In the adult the infection is generally considered to take place by way of the portal vein. Rolleston thinks that the infection takes place by way of the portal vein in chronic tuberculosis, and by way of the hepatic artery in acute tuberculosis. This view is also held by Zehden, who thinks that the initial foci are apt to be in the intermediate zone of the acini, where the lumen of the vessels is the narrowest. Orth[16] thinks that the infection travels from the portal vein by way of the lymph channels. Kotlar[17] made very careful examinations of the liver histologically and bacteriologically, and came to the conclusion that the infection was hematogenous and not via the bile ducts. Lancereaux[18] thinks that it comes from the intestine by way of the portal vein. Klebs[19] thinks with Zehden that the infection starts in the intermediate zone and spreads peripherally via the lymph channels. Gibbert and Lion[20] produced tuberculosis of the liver experimentally by injecting tubercle bacilli into the portal vein. The tubercles were found partially in the portal spaces and partially in the lobules.

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[10] Third Annual Report, Henry Phipps Institute, 1907.

[11] Semaine méd., 1891, No. 85.

[12] Revue de la Tuberculose, 1895.

[13] Acad. des sciences de l'Empereur Franz Joseph I, Bulletin International, Prague, 1895.

[14] Cntrlbl. für Bakt., 1900.

[15] Soc. de biologie, June 29, 1895.

Histological examination of 100 livers was made by us. The blocks were cut as for the routine histological work, no special care being taken to pick out pieces that seemed to contain tubercles. Of the 100 livers, miliary tubercles were found 79 times. Some of these lacked the giant cell, but the other characteristics were present, and we had no hesitancy in calling them tubercles. Seventy-four livers were stained for tubercle bacilli, with a positive result in 28, a percentage of 37.9. The infrequency with which tubercle bacilli are found in the liver has been commented upon by a number of observers, notably Baumgarten,[21] Brissard and Loupet,[22] Hanot and Lauth,[23] and Koeckel,[24] and we found that they are extremely hard to demonstrate. In two cases of acute miliary tuberculosis we found them in large numbers, but in all the rest we could only find a few, and then after a prolonged search.

The position of the tubercles was periportal 40 times, intracinal 17 times, and both periportal and intracinal 22 times. In a series of 45 examined particularly with reference to intestinal ulceration, the ulcers were found 38 times. Of these 38, the lesion in the liver was periportal 26 times, in 3 the lesion was intracinal, and in 9 the lesions were both periportal and intracinal. In the 7 cases showing no ulcers in the intestine, there were periportal tubercles in 2, intracinal in 3, and both periportal and intracinal in 4 cases. It seems likely from the findings in these cases that the infection generally comes from the intestine via the portal vein, but that in all probability it does come also from the general circulation by way of the hepatic artery. In two cases of acute miliary tuberculosis the tubercles were found in the portal spaces and also scattered throughout the parenchyma, and it is probable, as asserted by Zehden, that the infection in acute tuberculosis comes by way of the hepatic artery.

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[16] Virchow's Archiv, 1871, Band lxvi.

[17] Ztsch. für Heil., 1894, iv.

[18] Traité des Maladies des Foie et du Pancréas, 1899, 662.

[19] Allg. Path., 1869.

[20] Société de biologie, 1888.

[21] Ztschr. für klin. Med., Band ix, x.

[22] Études Exp., Sous la direct. du Prof. Verneuil, I, Paris, 1887.

[23] Ibid., II, Paris, 1888.

[24] Virchow's Archiv, 1896, Band cxliii.

II. SOLITARY TUBERCLE. Under this heading we include all the large caseous masses occurring in the liver, whether single or multiple. This is a very constant finding in the liver of many of the lower animals, particularly birds, where it is the only organ attacked in 20 per cent. According to Hutchinson,[25] tuberculosis of the liver was absent but twice in a series of 25 cases of avian tuberculosis. It occurs also very often in the liver of monkeys and cattle. Under this heading there are two subdivisions: (1) Conglomerate tubercles, and (2) tuberculous cavities or bile duct tubercles.

By *conglomerate tubercles* is meant the collection of large caseous masses which are found scattered throughout the liver substance. Craven Moore[26] reports a case of conglomerate tubercles in the liver in a case of cancer of the stomach, and thinks that the infection came from the ulcerated surface in the stomach. Orth reported two cases, but it was before the discovery of the tubercle bacillus, and the diagnosis was disputed by Zehden and Simmonds,[27] who think he had to do with either a gumma or a cancer. Other cases are reported by Simmonds, Rolleston, MacKenzie,[28] Anderson,[29] Middleton,[30] and Clement.[31] The centres of the nodules may undergo softening, in which event we may have the appearance of a cavity with caseating walls.

The *bile duct tubercles* are so called because of their association with the bile ducts, and it was claimed by Virchow that the ducts were occluded by tubercles and cysts formed. It has also been thought that the infection came from the intestine by way of the bile duct. Both these theories seem to be in error. Instead of the bile duct being occluded, it is likely that the tubercle caseates and breaks down, leaving a caseating cavity, which communicates with a bile-duct by extension and is oftentimes blood-or bile-stained, and this has given rise to the idea that the bile duct is occluded and forms a cyst.

As to the source of the infection, it has been claimed by Simmonds that it came by the bile duct, but Sargent,[32] Kotlar, and Sabourin[33] think that it comes by the portal vein or the hepatic artery, and that the duct is only secondarily involved by extension, the tubercle breaking into the duct and infecting it, and in this way producing other tubercles. Cases are reported by Simmonds, Hare,[34] Wethered,[35] and MacKenzie. In our series we have one case showing solitary

[25] Studies in Human and Comparative Pathology, p. 304.

[26] Medical Chronicle, October, 1889.

[27] Centralblatt für allgemeine Path., November, 1898.

[28] Transactions Path. Soc., xli, 146.

[29] The Australian Medical Gazette, March 20, 1899.

[30] Glasgow Journal, February, 1893.

[31] Virchow's Archiv, vol. cxxxix.

[32] Thèse de Paris, 1895.

[33] Le foie des tuberculeux, Archiv. de physiol. norm. et path. le 1er Juillet, 1883.

[34] Transactions London Path. Soc., 1858.

[35] Ibid., 1889.

tubercles associated with miliary tubercles. The lesions averaged about 1.5 cm. in diameter, there was a number present, but there was no cavity formation.

CIRRHOSIS. Cirrhosis of the liver may accompany tuberculosis and be due to an entirely different cause. For instance, we may have an alcoholic cirrhosis, superimposed upon a tuberculous infection. We may also have cirrhosis of the liver with tuberculosis elsewhere in the body, but it has long been felt that there is a fibrous hepatitis due to the tubercle bacillus. Briege, in 1879, described a fibrous hepatitis occurring in tuberculous patients, with the formation of fibrous tissue which enclosed the lobules, together with the newly formed bile ducts. Rolleston discusses at some length cirrhosis of the liver associated with tuberculosis. He quotes Hanot and Gilbert, who describe a large fatty liver with a small-cell infiltration and fibrous hyperplasia of the portal spaces, together with miliary tubercles. This they call "hypertrophic fatty tuberculous hepatitis."

These authors describe two other forms: (*a*) Cirrhosis without any enlargement of the liver; (*b*) cirrhosis with more fibrous tissue than the variety just mentioned, but with similar fatty change and tuberculous infiltration. The two latter forms differ only in the fact that one shows marked nodules, like those seen in cirrhosis with adenomas. Rolleston thinks that these forms occur, but he does not think them tuberculous. He considers it reasonable to suppose that the tubercle bacillus is capable of a sclerogenic effect on the liver. In animals, as in guinea-pigs, Hanot and Gilbert were able to produce a cirrhosis of the liver by the injection of avian tubercle bacilli, while human tubercle bacilli produced fatty change or fatty coagulation necrosis. Hanot found a deeply scarred liver, like that of acquired syphilis, associated with miliary tubercles in the organ. Collet and Gallivardin, according to Rolleston, report a case of tuberculosis of the portal spaces with a delicate fibrosis accompanying. Mixed infection, according to Rolleston, may set up a gastritis and a consequent dyspeptic type of cirrhosis. A marked grade of passive congestion may set up a cirrhosis—the so-called cardiac cirrhosis.

In this series one case of atrophic cirrhosis was found, and newly formed bile ducts and a slight increase of fibrous tissue several times; but the data are insufficient to warrant a conclusion that the tubercle bacillus is an etiological factor in these changes.

COINCIDENT PATHOLOGICAL PROCESSES. *Amyloid Degeneration.* This is a more or less frequent occurrence in chronic tuberculosis, not only of the lungs, but also of the bones. In this series it was found ten times, or 10 per cent.

Fatty change in the liver has been the subject of much discussion. Langerhans thinks that it is due to paralysis of the hepatic cells, and that the fat physiologically stored there is not given up. Klebs thinks that it is the direct result of the tuberculous process on the lymphatic apparatus of the liver, the lymph outflow being hindered and the fat retained. In our series it was present in 35 cases, or 35 per cent.

Congestion. The occurrence of congestion in the liver is, of course, not characteristic of this disease, and yet it is an almost constant finding. In chronic lung disease the embarassed respiratory apparatus, with its effect upon the right heart, sooner or later gives rise to passive congestion. The liver, with its close proximity and its blood supply, is one of the first organs to show the change. In our cases we classified the congestion as slight, moderate, and severe. There were 25 cases of slight congestion, 47 of moderate, and 22 of severe. In 6 cases congestion was not present.

CONCLUSIONS. 1. Miliary tubercles are found in the great majority of the livers of the cases of chronic phthisis autopsied.

2. Solitary tubercle of the liver is a very rare manifestation.

3. The infection probably is hematogenous, the bacilli being carried to the liver by the portal vein and the hepatic artery. In most cases the infection arises in intestinal ulcers.

4. Passive congestion of the liver is found in nearly every case of pulmonary tuberculosis, while amyloid and fatty change are found in a relatively small number of cases.

5. From our cases we are not convinced that a fibrosis or cirrhosis of the liver due to the tubercle bacilli does occur, and are more inclined to think that the fibroses found are due to other etiological factors.

My thanks are due to Dr. C. Y. White for the material on which this report is based.

ASCITES IN CIRRHOSIS OF THE LIVER CURED BY REPEATED TAPPINGS.

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In a paper widely read and frequently quoted, Hale White[1] has expressed himself in very pessimistic terms concerning ascites in cirrhosis. In studying the records of Guy's Hospital for five years, the works of Frerichs[2] and Murchison,[3] and a paper of Dr. F. Taylor,[4] he was unable to find a single patient with ascites due

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[1] Guy's Hospital Reports, 1893, xlix, 1.

[2] A Clinical Treatise on Diseases of the Liver. The Sydenham Society, London, 1861.

[3] Diseases of the Liver, 1867.

[4] Guy's Hospital Reports, xxxvi.